

09/968,720

=> fil reg

FILE 'REGISTRY' ENTERED AT 13:33:58 ON 22 JAN 2004

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STRUCTURE FILE UPDATES: 21 JAN 2004 HIGHEST RN 640234-51-1

DICTIONARY FILE UPDATES: 21 JAN 2004 HIGHEST RN 640234-51-1

TSCA INFORMATION NOW CURRENT THROUGH JULY 14, 2003

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Crossover limits have been increased. See HELP CROSSOVER for details.

Experimental and calculated property data are now available. For more
information enter HELP PROP at an arrow prompt in the file or refer
to the file summary sheet on the web at:

<http://www.cas.org/ONLINE/DBSS/registryss.html>

=> d his

(FILE 'HOME' ENTERED AT 12:55:42 ON 22 JAN 2004)

SET COST OFF

FILE 'REGISTRY' ENTERED AT 12:56:08 ON 22 JAN 2004

E MLYF/SQEP
L1 2 S E3
E MLFF/SQEP
L2 11 S E3
E MLFY/SQEP
L3 2 S E3
E C20H31N3O5S/MF
L4 28 S E3 AND 46.150.18/RID AND 1/NR
L5 4 S L4 AND LEUC? AND METHION?
L6 2 S L5 AND TYROS?
L7 1 S L6 NOT ISOLEUC?
L8 1 S L1 AND C29H40N4O6S
L9 1 S L3 AND C29H40N4O6S
L10 1 S L2 AND C29H40N4O5S
L11 4 S L7,L8,L9,L10
L12 12 S L1-L3 NOT L11
L13 6 S L12 AND 2/NR
L14 10 S L12 NOT MULTICHAIN/NTE
L15 10 S L13,L14

FILE 'HCAOLD' ENTERED AT 13:04:12 ON 22 JAN 2004

L16 0 S L11
L17 0 S L15

FILE 'USPATFULL, USPAT2' ENTERED AT 13:04:19 ON 22 JAN 2004

L18 1 S L11
L19 5 S L15
L20 6 S L18,L19

FILE 'HCAPLUS' ENTERED AT 13:04:47 ON 22 JAN 2004

L21 3 S L11
L22 32 S L15
L23 0 S L21 AND L22
L24 1 S US2002072499/PN OR WO2000-US7411/AP,PRN
E CLAGETT J/AU
L25 35 S E3-E7
E HISTATEK/PA,CS
L26 6 S E3-E12
L27 1 S L21,L24 AND L25,L26
L28 4 S L22 AND L24-L26
L29 7 S L21,L24,L27,L28
L30 26 S L22 AND (PD<=19990322 OR PRD<=19999322 OR
AD<=19990322)
L31 24578 S FIBROSIS
L32 40406 S ATHEROSCLER?
L33 17432 S CIRRHOS?
L34 2033 S GLOMERULOSCLERO?
L35 3135 S CHRONIC(L)PANCREAT?
L36 14089 S CORONARY(L)ARTER?(L)DISEASE
E FIBROSIS/CT
E E3+ALL
L37 7234 S E1+NT
E ATHEROSCLEROSIS/CT
E E3+ALL
L38 27457 S E10-E12,E9+NT
E E8+ALL
L39 31242 S E8+NT
E E14+ALL
L40 6716 S E4
E CIRRHOSIS/CT

	E E3+ALL
L41	10579 S E11+NT
	E GLOMERULOSCLEROSIS/CT
	E E3+ALL
L42	1040 S E2
	E CHRONIC PANCREATITIS/CT
	E E3+ALL
L43	461 S E2
	E CORONARY ARTERY DISEASE/CT
	E E3+ALL
L44	8573 S E2
	E PULMONARY FIBROSIS/CT
	E E3+ALL
L45	2087 S E2
	E TRAUMA/CT
	E E3+ALL
L46	2216 S E2
L47	16610 S TRAUM?
L48	39069 S SURGERY
L49	31434 S SURGICAL
L50	1 S L22 AND L31-L49
L51	1 S L21 AND L31-L49
L52	7 S L29,L50,L51
L53	23 S L30 NOT L52
	SEL DN AN 2
L54	1 S E1-E3
L55	8 S L52,L54 AND L21-L54
	E HISATEK/PA,CS
L56	1 S E3-E8 AND L55
L57	8 S L55,L56

09 | 968,720

FILE 'USPATFULL, USPAT2' ENTERED AT 13:27:26 ON 22 JAN 2004

FILE 'HCAPLUS' ENTERED AT 13:33:28 ON 22 JAN 2004

FILE 'REGISTRY' ENTERED AT 13:33:58 ON 22 JAN 2004

=> d sqide can tot l11

L11 ANSWER 1 OF 4 REGISTRY COPYRIGHT 2004 ACS on STN

RN 296233-40-4 REGISTRY

CN L-Tyrosine, L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

FS PROTEIN SEQUENCE; STEREOSEARCH

SQL 4

SEQ 1 MLFY

====

HITS AT: 1-4

RELATED SEQUENCES AVAILABLE WITH SEQLINK

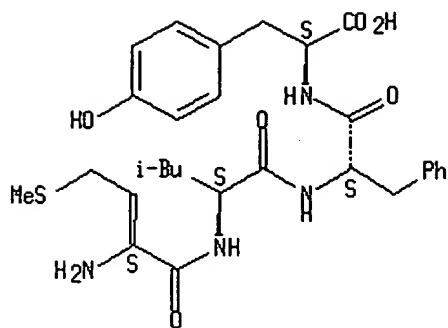
MF C29 H40 N4 O6 S

=====

SR CA

LC STN Files: CA, CAPLUS, USPATFULL

Absolute stereochemistry.



1 REFERENCES IN FILE CA (1907 TO DATE)

1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 133:247310

L11 ANSWER 2 OF 4 REGISTRY COPYRIGHT 2004 ACS on STN

RN 296233-39-1 REGISTRY

CN L-Phenylalanine, L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

FS PROTEIN SEQUENCE; STEREOSEARCH

SQL 4

SEQ 1 MLFF

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HITS AT: 1-4

RELATED SEQUENCES AVAILABLE WITH SEQLINK

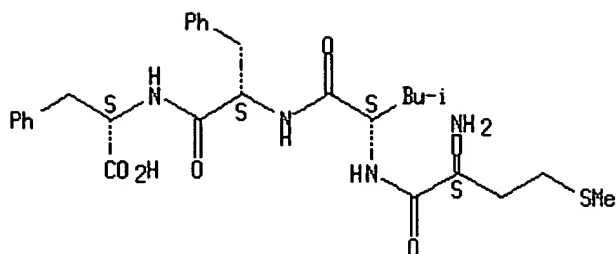
MF C29 H40 N4 O5 S

=====

SR CA

LC STN Files: CA, CAPLUS, USPATFULL

Absolute stereochemistry.



1 REFERENCES IN FILE CA (1907 TO DATE)

1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 133:247310

L11 ANSWER 3 OF 4 REGISTRY COPYRIGHT 2004 ACS on STN

RN 296233-38-0 REGISTRY

CN L-Phenylalanine, L-methionyl-L-leucyl-L-tyrosyl- (9CI) (CA INDEX NAME)

FS PROTEIN SEQUENCE; STEREOSEARCH

SQL 4

SEQ 1 MLYF

====

HITS AT: 1-4

RELATED SEQUENCES AVAILABLE WITH SEQLINK

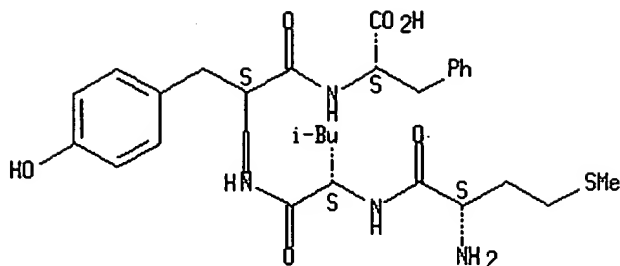
MF C29 H40 N4 O6 S

=====

SR CA

LC STN Files: CA, CAPLUS, USPATFULL

Absolute stereochemistry.



1 REFERENCES IN FILE CA (1907 TO DATE)

1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 133:247310

L11 ANSWER 4 OF 4 REGISTRY COPYRIGHT 2004 ACS on STN

RN 83613-43-8 REGISTRY

CN L-Tyrosine, L-methionyl-L-leucyl- (9CI) (CA INDEX NAME)

OTHER CA INDEX NAMES:

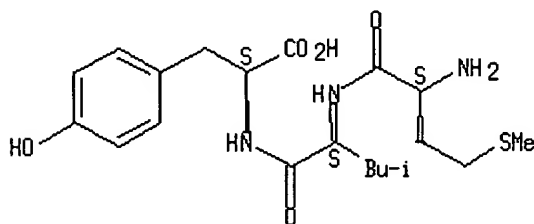
CN L-Tyrosine, N-(N-L-methionyl-L-leucyl)-

FS STEREOSEARCH

MF C20 H31 N3 O5 S

LC STN Files: CA, CAPLUS, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

3 REFERENCES IN FILE CA (1907 TO DATE)

3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 133:247310

REFERENCE 2: 114:24592

REFERENCE 3: 97:195014

=> fil hcaplus

FILE 'HCAPLUS' ENTERED AT 13:34:28 ON 22 JAN 2004

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FILE COVERS 1907 - 22 Jan 2004 VOL 140 ISS 4

FILE LAST UPDATED: 21 Jan 2004 (20040121/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d all hitstr tot l57

L57 ANSWER 1 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text	Citing References
--------------	----------------------

AN 2001:868244 HCAPLUS

DN 136:626

ED Entered STN: 30 Nov 2001

TI Modulation of alpha-6 integrin-mediated responses

IN Claggett, James; Lipani, John; Palmer, Craig

PA Histatek, LLC, USA

SO PCT Int. Appl., 58 pp.

CODEN: PIXXD2

DT Patent

LA English

IC ICM A61K038-04

ICS A61K038-06; A61K038-07; A61K038-17; A61K038-39; C07K005-00;

C07K005-08; C07K014-435; C07K014-705

CC 1-7 (Pharmacology)

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	<u>WO 2001089552</u>	A1	20011129	<u>WO 2001-US16774</u>	20010523
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W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM,
HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS,
LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO,
RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN,
YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1283715 A1 20030219 EP 2001-939365 20010523

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

US 2003050249 A1 20030313 US 2001-863837 20010523

BR 2001011083 A 20030408 BR 2001-11083 20010523

JP 2003534288 T2 20031118 JP 2001-585795 20010523

PRAI US 2000-206397P P 20000523

WO 2001-US16774 W 20010523

OS MARPAT 136:626

AB A method for modulating an alpha 6 subunit contg. integrin-mediated signal transduction is described. The method involves contacting a cell with an effective integrin modulating amt. of an alpha 6 subunit contg. integrin-mediated signal transduction pathway modification agent. Preferred agents are N-formyl peptides having the formula f-Met-Leu-X, wherein X is selected from the group consisting of Tyr, Tyr-Phe, Phe-Phe and Phe-Tyr. The method can be used to treat and VLA-6 integrin-mediated pathol. conditions such as the pro-inflammatory response, cancer metastasis or coronary heart disease.

ST alpha 6 integrin response modulation formyl peptide

IT Peptides, biological studies

RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);

THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(N-formyl; modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT Complement

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)

(activated fragment, pro-inflammatory activity of; modulation of
alpha-6 integrin-mediated responses to affect signal transduction and
treat pathol. conditions using agents such as N-formyl peptides)

IT T cell (lymphocyte)

(activated, modulation of interleukin α 6-contg.; modulation of
alpha-6 integrin-mediated responses to affect signal transduction and
treat pathol. conditions using agents such as N-formyl peptides)

IT Proteins

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(c-Raf, modulation of; modulation of alpha-6 integrin-mediated
responses to affect signal transduction and treat pathol. conditions
using agents such as N-formyl peptides)

IT Artery, disease

(coronary; modulation of alpha-6 integrin-mediated responses
to affect signal transduction and treat pathol. conditions using agents
such as N-formyl peptides)

IT G proteins (guanine nucleotide-binding proteins)

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(kinases for, modulation of; modulation of alpha-6 integrin-mediated
responses to affect signal transduction and treat pathol. conditions
using agents such as N-formyl peptides)

IT Antitumor agents

(metastasis; modulation of alpha-6 integrin-mediated responses to
affect signal transduction and treat pathol. conditions using agents
such as N-formyl peptides)

IT Anti-inflammatory agents

Antiasthmatics

Drug screening

Leukotriene antagonists

Signal transduction, biological

(modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT Formyl peptide receptors

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT Corticosteroids, biological studies

RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);

THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT Astrocyte

Basophil

Dendritic cell

Eosinophil

Lymphocyte

Macrophage

Mast cell

Mononuclear cell (leukocyte)

Polymorphonuclear leukocyte

(modulation of interleukin α 6-contg.; modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT Mitogens

(pro-inflammatory activity of; modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT Chemokines

Chemotactic factors

Cytokines

Interleukin 10

Interleukin 13

Interleukin 4

Interleukin 6

Interleukin 8

Tumor necrosis factors

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(pro-inflammatory activity of; modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT Integrins

RL: BSU (Biological study, unclassified); BIOL (Biological study)
($\alpha 6$; modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT Integrins

RL: BSU (Biological study, unclassified); BIOL (Biological study)
($\alpha 6 \beta 1$; modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT Adrenoceptor antagonists

($\beta 2$ -; modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT 80180-63-8

RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT 103171-49-9 115926-52-8, Phosphatidylinositol 3-kinase 137632-07-6,
ERK-1 kinase 139691-76-2, Gene c-Raf protein kinase 141588-29-6,
Kinase (phosphorylating), protein pp60c-src 372092-80-3, Protein kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(modulation of; modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT 59880-97-6 65154-06-5, Platelet activating factor 71160-24-2,
Leukotriene B4 80295-54-1, Complement C5a

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(pro-inflammatory activity of; modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

IT 63551-76-8, Phosphatidylinositide-specific phospholipase C

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(γ , protein kinase for, modulation of; modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Alexander; US 6017537 A 2000 HCAPLUS

(2) Roussel; J Leukocyte Biol 1997

(3) Wei; J Leukocyte Biol 1997, V61, P397 HCAPLUS

IT 80180-63-8

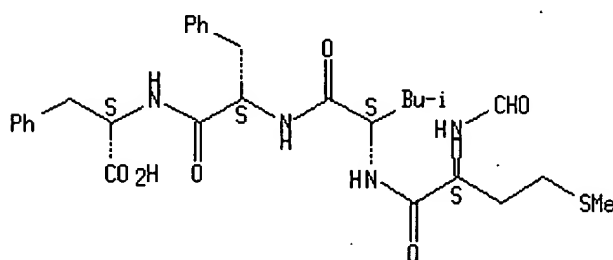
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(modulation of alpha-6 integrin-mediated responses to affect signal transduction and treat pathol. conditions using agents such as N-formyl peptides)

RN 80180-63-8 HCAPLUS

CN L-Phenylalanine, N-formyl-L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L57 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text	Citing References
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AN 2001:300744 HCAPLUS

DN 134:320856

ED Entered STN: 27 Apr 2001

TI N-Formyl peptide receptor complex with a G-protein kinase signal pathway
modification agent for inhibiting a pro-inflammatory response

IN Clagett, James A.; Palmer, Craig

PA Histatek, Llc, USA

SO PCT Int. Appl., 53 pp.

CODEN: PIXXD2

DT Patent

LA English

IC ICM C07K014-00

ICS G01N033-53

CC 1-7 (Pharmacology)

Section cross-reference(s): 6, 15

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	<u>WO 2001029069</u>	A1	20010426	<u>WO 2000-US28183</u> 20001012 <--
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W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP	<u>1222199</u>	A1	20020717	<u>EP 2000-970817</u> 20001012 <--
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R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL

BR	<u>2000014742</u>	A	20020827	<u>BR 2000-14742</u> 20001012 <--
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NO	<u>2002001732</u>	A	20020612	<u>NO 2002-1732</u> 20020412 <--
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PRAI	<u>US 1999-159677P</u>	P	19991015	<--
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	<u>WO 2000-US28183</u>	W	20001012	
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AB A method of inhibiting a pro-inflammatory response of a human peripheral blood mononuclear cell or polymorphonuclear cell, or fixed tissue cell is described. The cell is contacted with a pro-inflammatory agent to stimulate a pro-inflammatory response. Then, the cell is contacted with a G protein kinase signal pathway modification agent, thereby inhibiting inflammatory response signal transduction pathways mediated by G protein. A receptor complex is described wherein a G protein kinase signal pathway modification agent binds to a cell surface receptor of a human peripheral blood mononuclear cell or polymorphonuclear cell that has been stimulated

by a pro-inflammatory agent.

ST N formyl peptide receptor G protein signal modification; proinflammatory response inhibition G protein signal modification agent

IT Complement receptors

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(C5a, inhibition of; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Anti-inflammatory agents

Drug screening

Neutrophil

Signal transduction, biological

(N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Formyl peptide receptors

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)

(N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Ras proteins

RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)

(N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT G protein-coupled receptors

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT G proteins (guanine nucleotide-binding proteins)

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Complement

RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(activated fragment of, as proinflammatory agent; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT T cell (lymphocyte)

(activated, inhibition of proinflammatory response of, of human; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Interleukin 10

Interleukin 13

Interleukin 4

Interleukin 6

Interleukin 8

Tumor necrosis factors

RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(as proinflammatory agent; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Mitogens

(as proinflammatory agents; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Chemokines

Chemotactic factors

Cytokines

RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(as proinflammatory agents; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Receptors

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(complexes, with G protein kinase signal pathway modification agent; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Animal tissue

(fixed cell of, inhibiting proinflammatory response of human; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Mononuclear cell (leukocyte)

Polymorphonuclear leukocyte

(inhibiting proinflammatory response of human; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Astrocyte

Basophil

Dendritic cell

Eosinophil

Lymphocyte

Macrophage

Mast cell

Monocyte

(inhibition of proinflammatory response of, of human; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Chemokine receptors

Cytokine receptors

Immunoglobulin receptors

Interleukin 4 receptors

Interleukin 6 receptors

Interleukin 8 receptors

Platelet-activating factor receptors

Tumor necrosis factor receptors

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(inhibition of; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Interleukin receptors

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(interleukin 10 receptors, inhibition of; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for

inhibiting proinflammatory response)

IT Interleukin receptors

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(interleukin 13, inhibition of; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT Leukotriene receptors

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(leukotriene B4, inhibition of; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT 9026-43-1, Protein kinase 115926-52-8, Phosphatidylinositol 3-kinase 137632-07-6 139691-76-2, Raf-1 kinase 141349-89-5 141436-78-4, Protein kinase C 335632-58-1, G-Protein β -subunit kinase 335633-12-0, G-Protein α -subunit kinase

RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)

(N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT 11028-71-0, Concanavalin A

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT 80180-63-8 80180-63-8D, complexes

RL: BAC (Biological activity or effector, except adverse); BPR (Biological

process); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses) (as G protein kinase signal pathway modification agent; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT 18321-99-8D, peptides and complexes

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses) (as G protein kinase signal pathway modification agents; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT 65154-06-5, Platelet-activating factor 71160-24-2, Leukotriene B4

RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process) (as proinflammatory agent; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT 59880-97-6

RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process) (as proinflammatory agent; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

IT 27072-45-3D, FITC, HK-X conjugates

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(binding to activated receptors; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

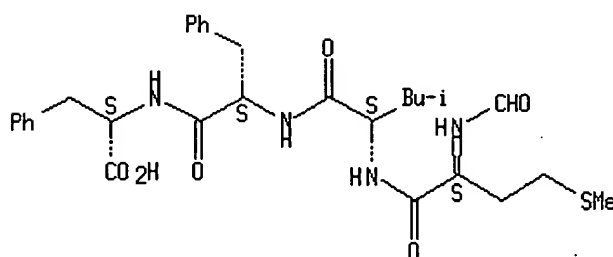
- (1) Bereta, J; Journal of Immunology 1992, V148(9), P2932 HCAPLUS
 - (2) Bevilacqua, M; European Journal of Pharmacology 1994, V283(3), P415
 - (3) Simon, J; Journal of Immunology 1991, V146(2), P476 HCAPLUS
 - (4) Smith, R; British Journal of Pharmacology 1995, V114(8), P1694 HCAPLUS
- IT 80180-63-8 80180-63-8D, complexes

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses) (as G protein kinase signal pathway modification agent; N-formyl peptide receptor complex with G-protein kinase signal pathway modification agent for inhibiting proinflammatory response)

RN 80180-63-8 HCAPLUS

CN L-Phenylalanine, N-formyl-L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

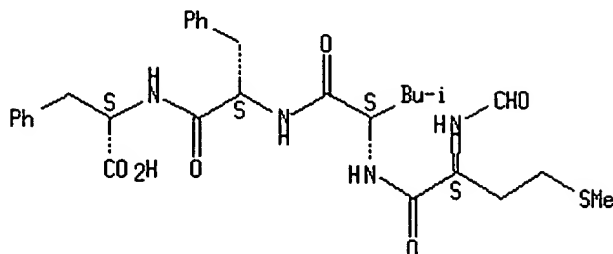
Absolute stereochemistry.



RN 80180-63-8 HCAPLUS

CN L-Phenylalanine, N-formyl-L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L57 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text	Citing References
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AN 2001:63842 HCAPLUS

DN 134:110460

ED Entered STN: 26 Jan 2001

TI Small peptides and methods using them for downregulation of IgE

IN Clargett, James

PA Histatek, LLC, USA

SO PCT.Int. Appl., 56 pp.

CODEN: PIXXD2

DT Patent

LA English

IC ICM A61K038-04

ICS A61K038-06; A61K038-07; C07K005-00; C07K005-08; C07K005-10

CC 1-7 (Pharmacology)

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	<u>WO 2001005420</u>	A1	20010125	<u>WO 2000-US19496</u>	20000714 <--
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W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,

CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL,

IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA,

MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI,
SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,
CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

BR 2000012495 A 20020611 BR 2000-12495 20000714 <--

EP 1303290 A1 20030423 EP 2000-950404 20000714 <--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, FI, CY

NO 2002000224 A 20020304 NO 2002-224 20020115 <--

PRAI US 1999-144539P P 19990716 <--

WO 2000-US19496 W 20000714

AB A method for downregulating IgE levels is described. The method involves administering to a patient an IgE-downregulating effective amt. of a peptide having the formula f-Met-Leu-X (X = Tyr, Tyr-Phe, Phe-Phe, Phe-Tyr).

ST IgE downregulation peptide

IT Glycoproteins, specific or class

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(CD40-L (antigen CD40 ligand); peptides for downregulating IgE)

IT Immunoglobulins

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(E; peptides for downregulating IgE)

IT Immunoglobulin receptors

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(IgE type I; peptides for downregulating IgE)

IT Immunoglobulin receptors

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(IgE type II, sol.; peptides for downregulating IgE)

IT Immunoglobulin receptors

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(IgE type II; peptides for downregulating IgE)

IT Immunoglobulin receptors

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(IgE; peptides for downregulating IgE)

IT Leukotrienes

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(anti-leukotrienes; peptides for downregulating IgE)

IT Antiasthmatics

Immunosuppressants

(peptides for downregulating IgE)

IT Corticosteroids, biological studies

Peptides, biological studies

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(peptides for downregulating IgE)

IT Lymphocyte

(plasma cell; peptides for downregulating IgE)

IT Adrenoceptor agonists

(β 2-; peptides for downregulating IgE)

IT 80180-63-8 97521-28-3 158724-27-7 225109-04-6

RL: BAC (Biological activity or effector, except adverse); BSU (Biological

study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)
(peptides for downregulating IgE)

IT 50-02-2, Dexamethasone

RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES
(Uses).

(peptides for downregulating IgE)

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS
RECORD

RE

(1) Hisatek Llc; WO 9925372 A1 1999 HCAPLUS

(2) Histatek Llc; WO 0032217 A1 2000 HCAPLUS

(3) Martens; US 4749685 A 1988 HCAPLUS

IT 80180-63-8 158724-27-7 225109-04-6

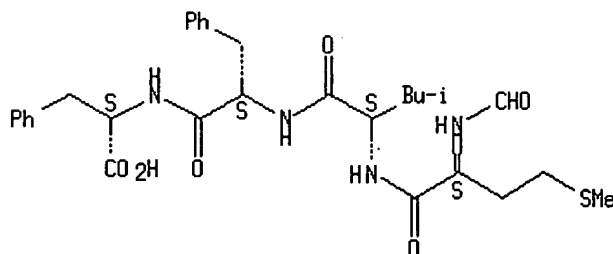
RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)

(peptides for downregulating IgE)

RN 80180-63-8 HCAPLUS

CN L-Phenylalanine, N-formyl-L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA
INDEX NAME)

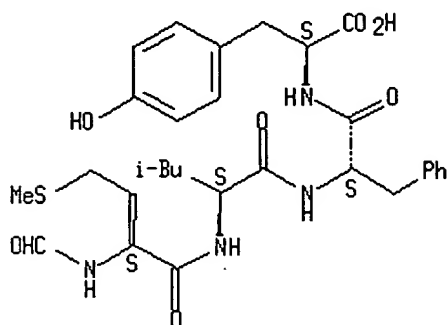
Absolute stereochemistry.



RN 158724-27-7 HCAPLUS

CN L-Tyrosine, N-formyl-L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

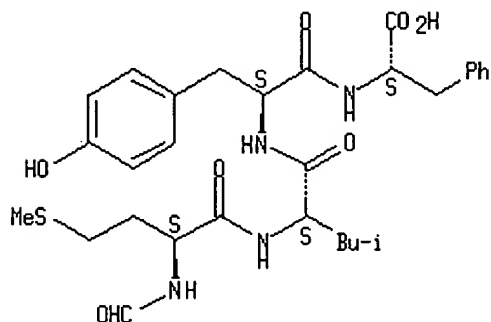
Absolute stereochemistry.



RN 225109-04-6 HCAPLUS

CN L-Phenylalanine, N-formyl-L-methionyl-L-leucyl-L-tyrosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L57 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text	Citing References
--------------	----------------------

AN 2000:688103 HCAPLUS

DN 133:247310

ED Entered STN: 29 Sep 2000

TI Treatment with small peptides to effect antifibrotic activity

IN Clagett, James

PA Histatek, LLC, USA

SO PCT Int. Appl., 44 pp.

CODEN: PIXXD2

DT Patent

LA English

IC ICM A61K038-00

ICS A61K038-05; A61K038-06; A61K038-07

CC 1-12 (Pharmacology)

FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 2000056349 A1 20000928 WO 2000-US7411 20000320 <--

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,
CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL,
IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA,
MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI,
SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1162990 A1 20011219 EP 2000-916561 20000320 <--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO

BR 2000009226 A 20011226 BR 2000-9226 20000320 <--

JP 2002539270 T2 20021119 JP 2000-606253 20000320 <--

NO 2001004594 A 20011121 NO 2001-4594 20010921 <--

US 2002072499 A1 20020613 US 2001-960720 20010921 <--

PRAI US 1999-125514P P 19990322

AB Methods for treating fibrosis in a mammal are described. An antifibrotic-effective amt. of a peptide f-Met-Leu-X (X = Tyr, Tyr-Phe, Phe-Phe, Phe-Tyr) is administered to the mammal. The fibrosis may be due to pathol. changes resulting, e.g., from pulmonary fibrosis, atherosclerosis, cirrhosis, glomerulosclerosis, chronic pancreatitis, coronary artery disease (such as caused by infection by bacterium Chlamydia pneumoniae , trauma or surgical procedures). Examples of surgical procedures that cause fibrosis are post-operative fibrosis peri-neurally in the dura or nerve roots following spinal surgery, tenolysis of injured or repaired tendons with adhesions, neurolysis of damaged or repaired peripheral nerves with adhesions, post-operative adhesions from gynecol. and abdominal surgeries, reparative surgery of the vas deferens or fallopian tubes for reversal of male or female sterilization, and surgical repair of other tubular structures such as urethra, intestine or esophagus.

ST peptide fibrosis treatment

IT Connective tissue

(adhesions; peptides for fibrosis treatment)

IT Asthma

(allergic, chronic; peptides for fibrosis treatment)

IT Biology

(anatomy, tubular structures, surgery for repair of; peptides for fibrosis treatment)

IT Pancreas, disease

(chronic pancreatitis, fibrosis from; peptides for fibrosis treatment)

IT Artery, disease

(coronary, fibrosis from; peptides for fibrosis treatment)

- IT Meninges
 - (dura mater, perineural post-operative fibrosis in; peptides for fibrosis treatment)
- IT Atherosclerosis
 - Surgery
 - (fibrosis from; peptides for fibrosis treatment)
- IT Lung, disease
 - (fibrosis; peptides for fibrosis treatment)
- IT Kidney, disease
 - (glomerulosclerosis, fibrosis from; peptides for fibrosis treatment)
- IT Disease, animal
 - (gynecol., surgery for; peptides for fibrosis treatment)
- IT Anti-inflammatory agents
 - Fibrosis
 - (peptides for fibrosis treatment)
- IT Peptides, biological studies
 - RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 - (peptides for fibrosis treatment)
- IT Nerve
 - (peripheral, neurolysis with adhesions; peptides for fibrosis treatment)
- IT Nerve
 - (root, perineural post-operative fibrosis in; peptides for fibrosis treatment)
- IT Nervous system
 - (sclerosis, fibrosis from; peptides for fibrosis treatment)

treatment)

IT Esophagus

Intestine

Urethra

(surgery for repair of; peptides for fibrosis treatment)

IT Sterility

(surgery for sterilization reversal; peptides for fibrosis treatment)

IT Spinal cord

(surgery, fibrosis from; peptides for fibrosis treatment)

IT Abdomen

Oviduct

Vas deferens

(surgery; peptides for fibrosis treatment)

IT Tendon

(tenolysis with adhesions; peptides for fibrosis treatment)

IT Injury

(trauma, fibrosis from; peptides for fibrosis treatment)

IT 83613-43-8 296233-38-0 296233-39-1

296233-40-4

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(peptides for fibrosis treatment)

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

- (1) Botha; Surgery 1995, V118, P358 MEDLINE
 (2) Lawrence; Clinical Experimental Immunology 1992, V89, P321 HCAPLUS
 (3) Macgregor, R; Alcoholism: Clinical and Experimental Research 1990, V14(2),
 P195 MEDLINE
 (4) Marceau, F; Biochemical Pharmacology 1996, V52(10), P1481 HCAPLUS
 (5) Sassen; Arteriosclerosis and Thrombosis 1993, V13(5), P651 HCAPLUS
 (6) Stragliotto; Arteriosclerosis and Thrombosis 1993, V13(6), P944 MEDLINE
 (7) Totani; Arteriosclerosis and Thrombosis 1994, V14(1), P125 HCAPLUS
 IT 83613-43-8 296233-38-0 296233-39-1

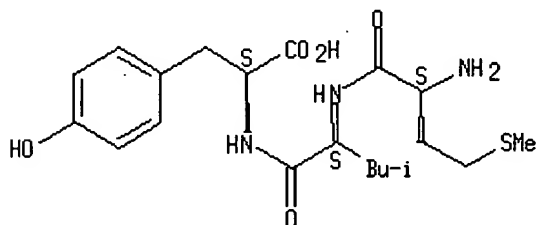
296233-40-4

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (peptides for fibrosis treatment)

RN 83613-43-8 HCAPLUS

CN L-Tyrosine, L-methionyl-L-leucyl- (9CI) (CA INDEX NAME)

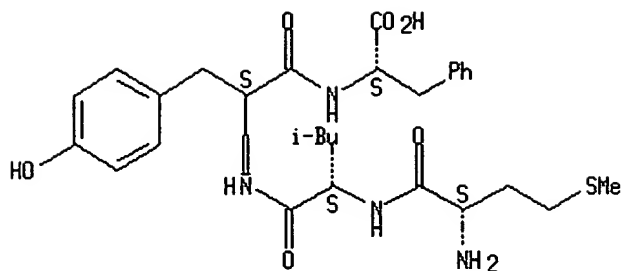
Absolute stereochemistry.



RN 296233-38-0 HCAPLUS

CN L-Phenylalanine, L-methionyl-L-leucyl-L-tyrosyl- (9CI) (CA INDEX NAME)

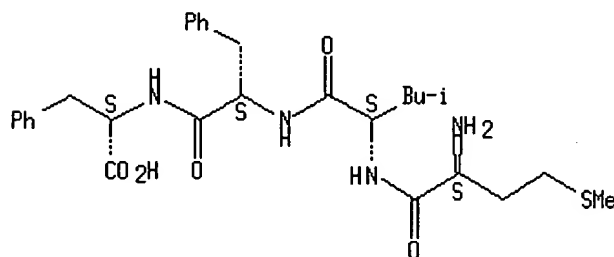
Absolute stereochemistry.



RN 296233-39-1 HCAPLUS

CN L-Phenylalanine, L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

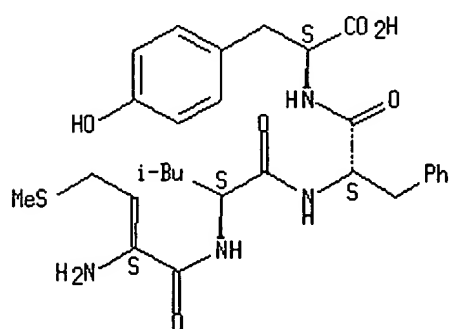
Absolute stereochemistry.



RN 296233-40-4 HCAPLUS

CN L-Tyrosine, L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L57 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text	Citing References
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AN 2000:383954 HCAPLUS

DN 133:26852

ED Entered STN: 09 Jun 2000

TI Small peptides and methods using them for treatment of asthma and
inflammation

IN Houck, John C.; Clagett, James

PA Histatek, LLC, USA

SO PCT Int. Appl., 74 pp.

CODEN: PIXXD2

DT Patent

LA English

IC ICM A61K038-07

ICS A61K038-06

CC 1-7 (Pharmacology)

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	<u>WO 2000032217</u>	A1	20000608	<u>WO 1998-US25583</u>	19981203 <--
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W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG,
KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX,
NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT,
UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,
ES,

FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI,
CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

<u>AU 9918018</u>	A1	20000619	<u>AU 1999-18018</u>	19981203 <--
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<u>EP 1152770</u>	A1	20011114	<u>EP 1998-962874</u>	19981203 <--
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R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,

IE, FI

BR 9816097 A 20020122 BR 1998-16097 19981203 <--

JP 2003504304 T2 20030204 JP 2000-584908 19981203 <--

PRAI WO 1998-US25583 A 19981203 <--

AB Methods for treating allergies, cutaneous inflammation, arthritis, chronic obstruction pulmonary disease and treating chronic inflammatory bowel disease are described. Also described is a method for inhibiting the infiltration of eosinophils into airways of a patient, a method for inhibiting the mucous release into airways of a patient, a method for blocking IgE activation of a lymphocyte, a method for stabilizing the cell membrane of a lymphocyte, thereby preventing their further involvement in the increased inflammatory response to an IgE antigen challenge, and a method for inhibiting the migration of T-cells. These methods involve administering to the patient a therapeutically effective amt. of a peptide having the formula f-Met-Leu-X, (X = Tyr, Tyr-Phe, Phe-Phe, Phe-Tyr).

ST asthma inflammation allergy arthritis treatment peptide

IT Immunoglobulins

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(E; peptides for treatment of asthma and inflammation)

IT Cell migration

(T-cell; peptides for treatment of asthma and inflammation)

IT Lymphocyte

Macrophage

Monocyte

Neutrophil

(activation; peptides for treatment of asthma and inflammation)

IT Eosinophil

(airways infiltration; peptides for treatment of asthma and

inflammation)

IT Nose

(allergic rhinitis; peptides for treatment of asthma and inflammation)

IT Leukotrienes

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(anti-leukotrienes; peptides for treatment of asthma and inflammation,
and use with other agents)

IT Lung, disease

(chronic obstructive; peptides for treatment of asthma and
inflammation)

IT Dermatitis

(contact; peptides for treatment of asthma and inflammation)

IT Mast cell

(degranulation; peptides for treatment of asthma and inflammation)

IT Respiratory tract

(eosinophil infiltration; peptides for treatment of asthma and
inflammation)

IT Arthritis

(including spondylarthritis; peptides for treatment of asthma and
inflammation)

IT Respiratory tract

(inflammation; peptides for treatment of asthma and inflammation)

IT Intestine, disease

(inflammatory, chronic; peptides for treatment of asthma and
inflammation)

IT Cell membrane

(lymphocyte, stabilization; peptides for treatment of asthma and
inflammation)

IT Cell activation

(lymphocyte; peptides for treatment of asthma and inflammation)

- IT Cell activation
 - (macrophage; peptides for treatment of asthma and inflammation)
- IT Cell degranulation
 - (mast cell; peptides for treatment of asthma and inflammation)
- IT CD4-positive T cell
 - T cell (lymphocyte)
 - (migration; peptides for treatment of asthma and inflammation)
- IT Cell activation
 - (monocyte; peptides for treatment of asthma and inflammation)
- IT Cell activation
 - (neutrophil; peptides for treatment of asthma and inflammation)
- IT Aging, animal
 - Allergy inhibitors
 - Anti-inflammatory agents
 - Antiarthritics
 - Antiasthmatics
 - Dermatitis
 - Drug allergy
 - Eczema
 - Food allergy
 - Lupus erythematosus
 - Psoriasis
 - Sunburn
 - Urticaria
 - (peptides for treatment of asthma and inflammation)
- IT Peptides, biological studies
 - RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 - (peptides for treatment of asthma and inflammation)

IT Tumor necrosis factors

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(peptides for treatment of asthma and inflammation)

IT Corticosteroids, biological studies

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(peptides for treatment of asthma and inflammation, and use with other agents)

IT Arthritis

(psoriatic arthritis; peptides for treatment of asthma and inflammation)

IT Mucus

(release into airways; peptides for treatment of asthma and inflammation)

IT Adrenoceptor agonists

(β 2-; peptides for treatment of asthma and inflammation, and use with other agents)

IT 111406-87-2, Zileuton

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(peptides for treatment of asthma and inflammation)

IT 59880-97-6 65929-03-5 67247-11-4 67247-12-5 73572-34-6
80180-62-7 105931-59-7 225109-05-7

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(peptides for treatment of asthma and inflammation)

IT 80180-63-8 97521-28-3 158724-27-7 225109-04-6

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL

(Biological study); USES (Uses)

(peptides for treatment of asthma and inflammation)

IT 80619-02-9, 5-Lipoxygenase

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL

(Biological study); PROC (Process)

(peptides for treatment of asthma and inflammation)

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS
RECORD

RE

(1) Duff, R; US 5776892 A 1998 HCAPLUS

(2) Gen Hospital Corp; EP 0398143 A 1990 HCAPLUS

(3) Hisatek, L; WO 9925372 A 1999 HCAPLUS

IT 80180-63-8 158724-27-7 225109-04-6

RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL

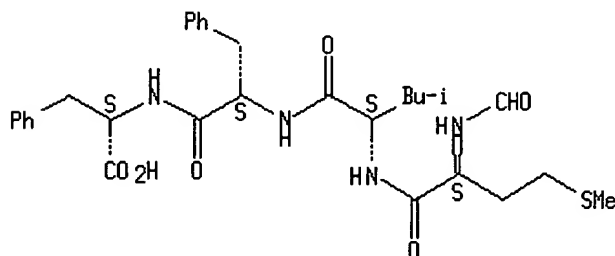
(Biological study); USES (Uses)

(peptides for treatment of asthma and inflammation)

RN 80180-63-8 HCAPLUS

CN L-Phenylalanine, N-formyl-L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA
INDEX NAME)

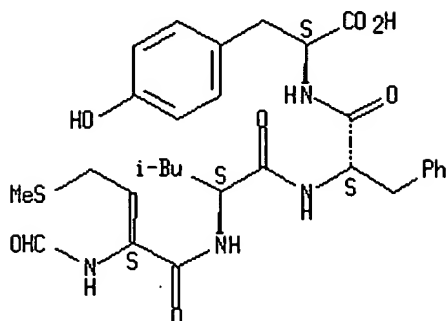
Absolute stereochemistry.



RN 158724-27-7 HCAPLUS

CN L-Tyrosine, N-formyl-L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX
NAME)

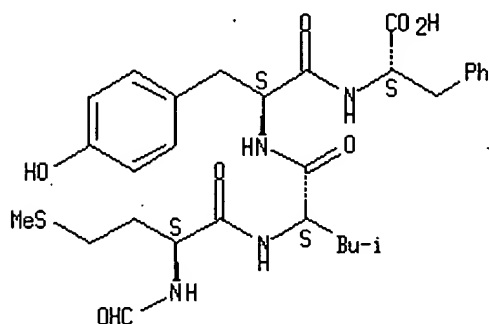
Absolute stereochemistry.



RN 225109-04-6 HCAPLUS

CN L-Phenylalanine, N-formyl-L-methionyl-L-leucyl-L-tyrosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L57 ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text	Citing References
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AN 1999:350603 HCAPLUS

DN 130:347411

ED Entered STN: 08 Jun 1999

TI Small peptides and methods for treatment of asthma and inflammation

IN Houck, John C.

PA Hisatek, LLC, USA

SO PCT Int. Appl., 48 pp.

CODEN: PIXXD2

DT Patent

LA English

IC ICM A61K038-06

ICS A61K038-07; A61K039-02; C07K005-00; C07K005-08; C07K005-10

CC 1-7 (Pharmacology)

Section cross-reference(s): 63

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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<u>PI WO 9925372</u>	A1	19990527	<u>WO 1998-US14103</u>	19980707 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,				

FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

<u>CA 2309639</u>	AA	19990527	<u>CA 1998-2309639</u>	19980707 <--
<u>AU 9884779</u>	A1	19990607	<u>AU 1998-84779</u>	19980707 <--
<u>EP 1037651</u>	A1	20000927	<u>EP 1998-935561</u>	19980707 <--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

<u>BR 9815288</u>	A	20010213	<u>BR 1998-15288</u>	19980707 <--
<u>JP 2002516820</u>	T2	20020611	<u>JP 2000-520805</u>	19980707 <--
<u>US 6391856</u>	B1	20020521	<u>US 1998-190043</u>	19981110 <--
<u>US 6462020</u>	B1	20021008	<u>US 1998-189130</u>	19981110 <--

US 2003013658 A1 20030116 US 2002-147633 20020516 <--
US 2003130200 A1 20030710 US 2002-192000 20020709 <--
PRAI US 1997-65336P P 19971113 <--
WO 1998-US14103 W 19980707 <--
US 1998-189130 A1 19981110 <--
US 1998-190043 A3 19981110 <--

AB A pharmaceutical compn. is described as an admixt. of a pharmacol. carrier and a peptide having the formula f-Met-Leu-X (X = Tyr, Tyr-Phe, Phe-Phe and Phe-Tyr). Also described are methods for inhibiting the degranulation of mast cells and for treating inflammation in a patient, for example, where the inflammation is a result of a disease selected from the group consisting of asthma, rheumatoid arthritis and anaphylaxis. In addn., methods are described for inhibiting the release of cytokines in a patient, for inhibiting the release of histamines in a patient, for inhibiting the release leukotrienes in a patient, for reducing adhesion, migration and aggregation of lymphocytes, eosinophils and neutrophils to a site of inflammation in a patient, for reducing the prodn. of IgE antibodies at site of inflammation in a patient, and for inhibiting increased vascular permeability at site of inflammation in a patient. The methods use the described pharmaceutical compn.

ST peptide antiinflammatory asthma inhibitor; mast cell degranulation inhibitor peptide; cytokine histamine leukotriene release inhibitor peptide; IgE vascular permeability inhibition peptide

IT Immunoglobulins

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(E; peptides and methods for treatment of asthma and inflammation)

IT Crosslinking

(IgE; peptides and methods for treatment of asthma and inflammation)

IT Antibodies

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(IgE; peptides and methods for treatment of asthma and inflammation)

IT Eosinophil

(adhesion, migration, and aggregation; peptides and methods for treatment of asthma and inflammation)

IT Lymphocyte

Neutrophil

(adhesion; peptides and methods for treatment of asthma and inflammation)

IT Lymphocyte

(aggregation; peptides and methods for treatment of asthma and inflammation)

IT Mast cell

(degranulation, inhibitors; peptides and methods for treatment of asthma and inflammation)

IT Drug delivery systems

(inhalants; peptides and methods for treatment of asthma and inflammation)

IT Cell adhesion

Cell migration

(lymphocyte; peptides and methods for treatment of asthma and inflammation)

IT Cell degranulation

(mast cell, inhibitors; peptides and methods for treatment of asthma and inflammation)

IT Neutrophil

(migration and aggregation; peptides and methods for treatment of asthma and inflammation)

IT Lymphocyte

(migration; peptides and methods for treatment of asthma and inflammation)

IT Cell adhesion

(neutrophil; peptides and methods for treatment of asthma and inflammation)

IT Drug delivery systems

(oral; peptides and methods for treatment of asthma and inflammation)

IT Anaphylaxis

Anti-inflammatory agents

Antiasthmatics

Antihistamines

Antirheumatic agents

Drug delivery systems

(peptides and methods for treatment of asthma and inflammation)

IT Corticosteroids, biological studies

Peptides, biological studies

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(peptides and methods for treatment of asthma and inflammation)

IT Cytokines

Leukotrienes

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(peptides and methods for treatment of asthma and inflammation)

IT Blood vessel

(permeability; peptides and methods for treatment of asthma and inflammation)

IT Biological transport

(permeation, vascular; peptides and methods for treatment of asthma and

inflammation)

IT Drug delivery systems

(sprays; peptides and methods for treatment of asthma and inflammation)

IT Drug delivery systems

(tablets; peptides and methods for treatment of asthma and inflammation)

IT Drug delivery systems

(topical; peptides and methods for treatment of asthma and inflammation)

IT Adrenoceptor agonists

(β 2-; peptides and methods for treatment of asthma and inflammation)

IT 65929-03-5 67247-11-4 67247-12-5 73572-34-6 80180-62-7
225109-05-7 225111-44-4

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)

(peptides and methods for treatment of asthma and inflammation)

IT 80180-63-8 97521-28-3 158724-27-7 225109-04-6

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(peptides and methods for treatment of asthma and inflammation)

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Abe; US 4929623 A 1990 HCAPLUS

(2) Anderson, R; Digestive Diseases and Sciences 1993, V27(2), P248

(3) Anon; Goodman and Gilman's, "The Pharmacological Basis of Therapeutics" 1980, P170

(4) Casale, T; Annals of Allergy 1983, V51(1 Part 1), P2

(5) Ferry, D; Gastroenterology 1989, V97, P61 HCAPLUS

(6) Gleisner, J; Inflammation 1981, V5(1), P13 HCAPLUS

(7) Kermode, J; Biochemistry Journal 1991, V276, P715 HCAPLUS

(8) Kuna, P; The Journal of Experimental Medicine 1992, V175, P489 HCAPLUS

IT 80180-63-8 158724-27-7 225109-04-6

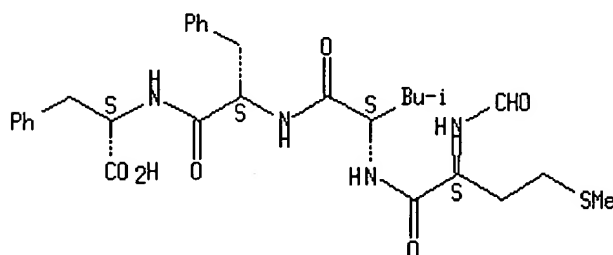
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(peptides and methods for treatment of asthma and inflammation)

RN 80180-63-8 HCAPLUS

CN L-Phenylalanine, N-formyl-L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

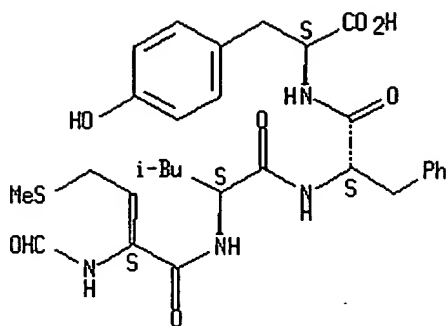
Absolute stereochemistry.



RN 158724-27-7 HCAPLUS

CN L-Tyrosine, N-formyl-L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

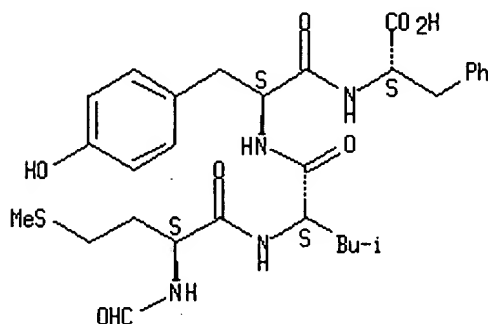
Absolute stereochemistry.



RN 225109-04-6 HCAPLUS

CN L-Phenylalanine, N-formyl-L-methionyl-L-leucyl-L-tyrosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L57 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text	Citing References
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AN 1991:24592 HCAPLUS

DN 114:24592

ED Entered STN: 26 Jan 1991

TI Capillary electrophoretic separations of peptides using micelle-forming compounds and cyclodextrins as additives

AU Liu, Jinping; Cobb, Kelly A.; Novotny, Milos

CS Dep. Chem., Indiana Univ., Bloomington, IN, 47405, USA

SO Journal of Chromatography (1990), 519(1), 189-97

CODEN: JOGRAM; ISSN: 0021-9673

DT Journal

LA English

CC 34-3 (Amino Acids, Peptides, and Proteins)

Section cross-reference(s): 80

AB The value of electrokinetic capillary chromatog. for sepg. structurally similar model peptides and tryptic digests is demonstrated. The behavior of model peptides in buffer systems contg. dodecyltrimethylammonium bromide, hexadecyltrimethylammonium bromide, sodium dodecyl sulfate, and

2

cyclodextrins as additives is described. These additives, under different anal. circumstances, exhibit certain beneficial effects for peptides with similar net charges but different hydrophobicities. Sepns. of underivatized peptides, utilizing UV detection, are presented. In addn., sepn. of fluorescent products of peptides derivatized with o-phthalaldehyde, fluorescamine, and a new reagent, 3-(4-carboxybenzoyl)-2-quinolinecarboxaldehyde, are demonstrated and discussed. Beneficial spectroscopic detection effects with cyclodextrin are also noted.

ST capillary zone electrophoresis peptide surfactant; cyclodextrin peptide capillary zone electrophoresis; micelle peptide capillary zone electrophoresis; fluorescence carboxybenzoylquinolinecarboxaldehyde peptide adduct; quinolinecarboxaldehyde carboxybenzoyl peptide fluorescence probe

IT Peptides, analysis

RL: ANT (Analyte); ANST (Analytical study)

(sepn. of, by capillary zone electrophoresis using micelle-forming compds. and cyclodextrins)

IT Fluorescent substances

(probes, (carboxybenzoyl)quinolinecarboxaldehyde as, for peptides)

IT Electrophoresis and Ionophoresis

(zone, capillary, of peptides using micelle-forming compds. and

cyclodextrins)

IT 57-09-0, Hexadecyltrimethylammonium bromide 151-21-3, Sodium dodecylsulfate, uses and miscellaneous 1119-94-4,

Dodecyltrimethylammonium bromide 7585-39-9, β -Cyclodextrin 10016-20-3, α -Cyclodextrin

RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses) (additive, for capillary zone electrophoresis of peptides)

IT 643-79-8, o-Phthalaldehyde 38183-12-9, Fluorescamine 131124-59-9

RL: RCT (Reactant); RACT (Reactant or reagent)

(derivatization by, of peptides in capillary zone electrophoresis)

IT 53-73-6 58-49-1 484-42-4 4306-24-5 4474-91-3 13602-53-4, Angiotensin III 16376-83-3 21957-32-4 31461-61-7 42155-93-1 52498-25-6 56317-01-2 59881-08-2 61756-22-7 61756-28-3 70195-20-9 83613-43-8 99624-52-9

RL: ANT (Analyte); ANST (Analytical study)

(sepn. of, from mixt. by capillary zone electrophoresis using micelle-forming compds. and cyclodextrins)

IT 83613-43-8

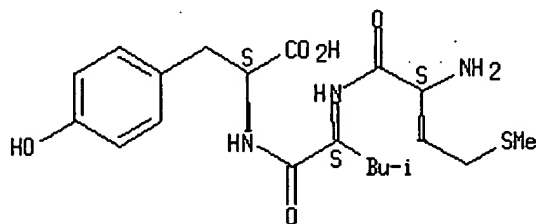
RL: ANT (Analyte); ANST (Analytical study)

(sepn. of, from mixt. by capillary zone electrophoresis using micelle-forming compds. and cyclodextrins)

RN 83613-43-8 HCAPLUS

CN L-Tyrosine, L-methionyl-L-leucyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



Full Text	Citing References
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AN 1982:595014 HCAPLUS

DN 97:195014

ED Entered STN: 12 May 1984

TI A highly specific aminotripeptidase of rat brain cytosol. Substrate
specificity and effects of inhibitors

AU Sachs, Len; Marks, Neville

CS Cent. Neurochem., Rockland Res. Inst., Ward's Island, NY, 10035, USA

SO Biochimica et Biophysica Acta (1982), 706(2), 229-38

CODEN: BBACAQ; ISSN: 0006-3002

DT Journal

LA English

CC 7-3 (Enzymes)

AB An aminopeptidase preferentially hydrolyzing Leu-Gly-Gly or Ala-Gly-Gly was purified from rat brain cytosol and its substrate specificity and the effects of inhibitors investigated. The enzyme was devoid of di- and oligopeptidase contamination. Biol. active tripeptides such as Met-Leu-Tyr (chemotactic factor), Gly-His-Lys (liver growth factor), and Thr-Val-Leu (central nervous system tripeptide) were hydrolyzed at rates 0.05-0.15-fold that of Leu-Gly-Gly. Melanostatin (Pro-Leu-GlyNH₂) was not a substrate. Substrates bearing N-terminal charged groups, substrates with proline in positions 2 or 3, those with a D-amino acid in positions 1 or 2, or with a C-terminal CONH₂ were poorly hydrolyzed or did not act as substrates, thus providing information on subsites involved in enzyme catalysis. The enzyme was inhibited competitively by bestatin ($K_i = 10^{-7}M$) and Captopril ($2.5 \times 10^{-7}M$). Inhibition occurred with low concns. of Zn²⁺ or p-chloromercuribenzoate, and, at higher concn., with L-1-tosyl-phenylalanylchloromethyl ketone and p-

chloromercuriphenylsulfonate. Inhibition was obsd. for the chemotactic factor ($I_{50} = 13 \mu\text{M}$) and for the central nervous system tripeptide ($195 \mu\text{M}$). The enhanced action of Captopril was attributed to the presence of SH and Me groups, as inhibition was shared by di- and tripeptides with proline in positions 2 and 3. The specificity pattern of the brain enzyme was different from that reported for kidney and intestine.

ST aminotripeptidase brain substrate specificity inhibitor; cytosol aminotripeptidase brain specificity; peptide specificity aminotripeptidase brain

IT Brain, composition

(aminotripeptidase of, substrate specificity and inhibitors of)

IT Michaelis constant

(of aminotripeptidase)

IT Kinetics, enzymic

(of inhibition, of aminotripeptidase)

IT Molecular structure-biological activity relationship

(aminotripeptidase-inhibiting, of tripeptides)

IT Cytoplasm

(cytosol, aminotripeptidase of, of brain, substrate specificity and inhibitors of)

IT Peptides, biological studies

RL: BIOL (Biological study)

(tri-, aminotripeptidase of brain cytosol specificity for)

IT 61-90-5, biological studies 64-69-7 66-71-7 128-53-0 328-38-1
329-98-6 402-71-1 554-77-8 556-50-3 704-15-4 7440-66-6,
biological studies 9076-44-2 13184-14-0 23815-91-0 37691-11-5
58569-55-4 59880-97-6

RL: BIOL (Biological study)

(aminotripeptidase of brain cytosol inhibition by)

IT 58970-76-6 62571-86-2

RL: BIOL (Biological study)

(aminotripeptidase of brain cytosol inhibition by, kinetics of)

IT 70-18-8, biological studies 556-33-2 926-79-4 1187-50-4 1948-31-8
2002-44-0 2576-67-2 3146-40-5 5874-90-8 6234-26-0 6620-98-0
6745-19-3 7561-25-3 10329-75-6 14486-08-9 14857-82-0 18625-22-4
19245-85-3 19408-48-1 21778-69-8 23576-41-2 23576-42-3
30802-27-8 32999-80-7 36930-55-9 38678-77-2 42155-93-1
42538-53-4 49557-75-7 52027-85-7 55488-08-9 66317-22-4
83613-43-8

RL: BIOL (Biological study)

(aminotripeptidase of brain cytosol specificity for)

IT 9056-26-2

RL: PROC (Process)

(of brain cytosol, substrate specificity and inhibition of)

IT 83613-43-8

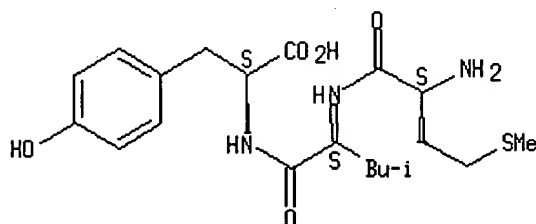
RL: BIOL (Biological study)

(aminotripeptidase of brain cytosol specificity for)

RN 83613-43-8 HCAPLUS

CN L-Tyrosine, L-methionyl-L-leucyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



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FILE 'USPATFULL' ENTERED AT 13:35:08 ON 22 JAN 2004

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L18 ANSWER 1 OF 1 USPATFULL on STN

Full Text	Citing References
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AN 2002:141513 USPATFULL

TI Treatment with small peptides to effect antifibrotic activity

IN Clagett, James, Snohomish, WA, UNITED STATES

PA Histatek, Inc. (U.S. corporation)

PI US 2002072499 A1. 20020613

AI US 2001-960720 A1 20010921 (9)

RLI Continuation of Ser. No. WO 2000-US7411, filed on 20 Mar 2000,
UNKNOWN

PRAI US 1999-125514P 19990322 (60)

DT Utility

FS APPLICATION

LREP Edwards & Angell, LLP, P.O. Box 9169, Boston, MA, 02209

CLMN Number of Claims: 12

ECL Exemplary Claim: 1

DRWN 13 Drawing Page(s)

LN.CNT 814

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods for treating treating fibrosis in a mammal are described. An
antifibrotic effective amount of a peptide having the formula
f-Met-Leu-X where X is selected from the group consisting of Tyr,
Tyr-Phe, Phe-Phe and Phe-Tyr is administered to the mammal. The fibrosis

may be due to pathological changes resulting, e.g., from pulmonary fibrosis, atherosclerosis, cirrhosis, glomerulosclerosis, chronic pancreatitis, coronary artery disease (such as caused by infection by bacterium *Chlamydia pneumoniae*), trauma or surgical procedures.

Examples

of surgical procedures that cause fibrosis are post-operative fibrosis peri-neurally in the dura or nerve roots following spinal surgery, tenolysis of injured or repaired tendons with adhesions, neurolysis of damaged or repaired peripheral nerves with adhesions, post-operative adhesions from gynecologic and abdominal surgeries, reparative surgery of the vas deferens or fallopian tubes for reversal of male or female sterilization, and surgical repair of other tubular structures such as urethra, intestine or esophagus.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

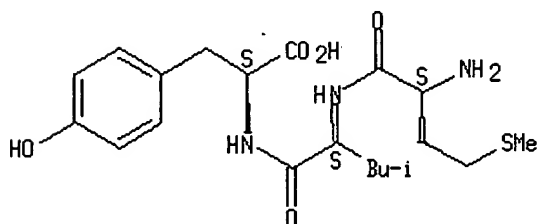
IT 83613-43-8 296233-38-0 296233-39-1
296233-40-4

(peptides for fibrosis treatment)

RN 83613-43-8 USPATFULL

CN L-Tyrosine, L-methionyl-L-leucyl- (9CI) (CA INDEX NAME)

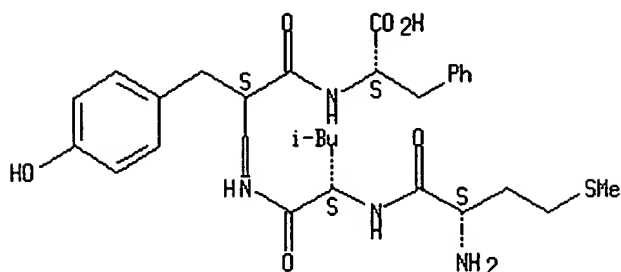
Absolute stereochemistry.



RN 296233-38-0 USPATFULL

CN L-Phenylalanine, L-methionyl-L-leucyl-L-tyrosyl- (9CI) (CA INDEX NAME)

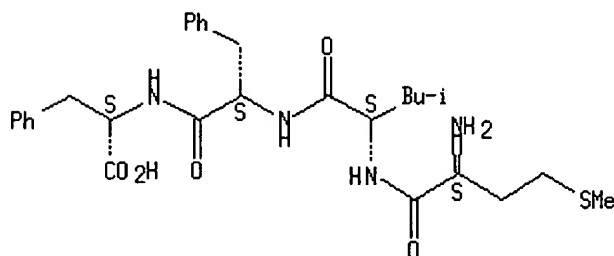
Absolute stereochemistry.



RN 296233-39-1 USPATFULL

CN L-Phenylalanine, L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 296233-40-4 USPATFULL

CN L-Tyrosine, L-methionyl-L-leucyl-L-phenylalanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

